Respiratory Failure

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RESPIRATORY FAILURE

- "inability of the lung to meet the metabolic demands of the body. This can be from failure of tissue oxygenation and/or failure of CO_2 homeostasis."

RESPIRATORY FAILURE

- Definition

  Respiration is gas exchange between the organism and its environment. Function of respiratory system is to transfer O_2 from atmosphere to blood and remove CO_2 from blood.

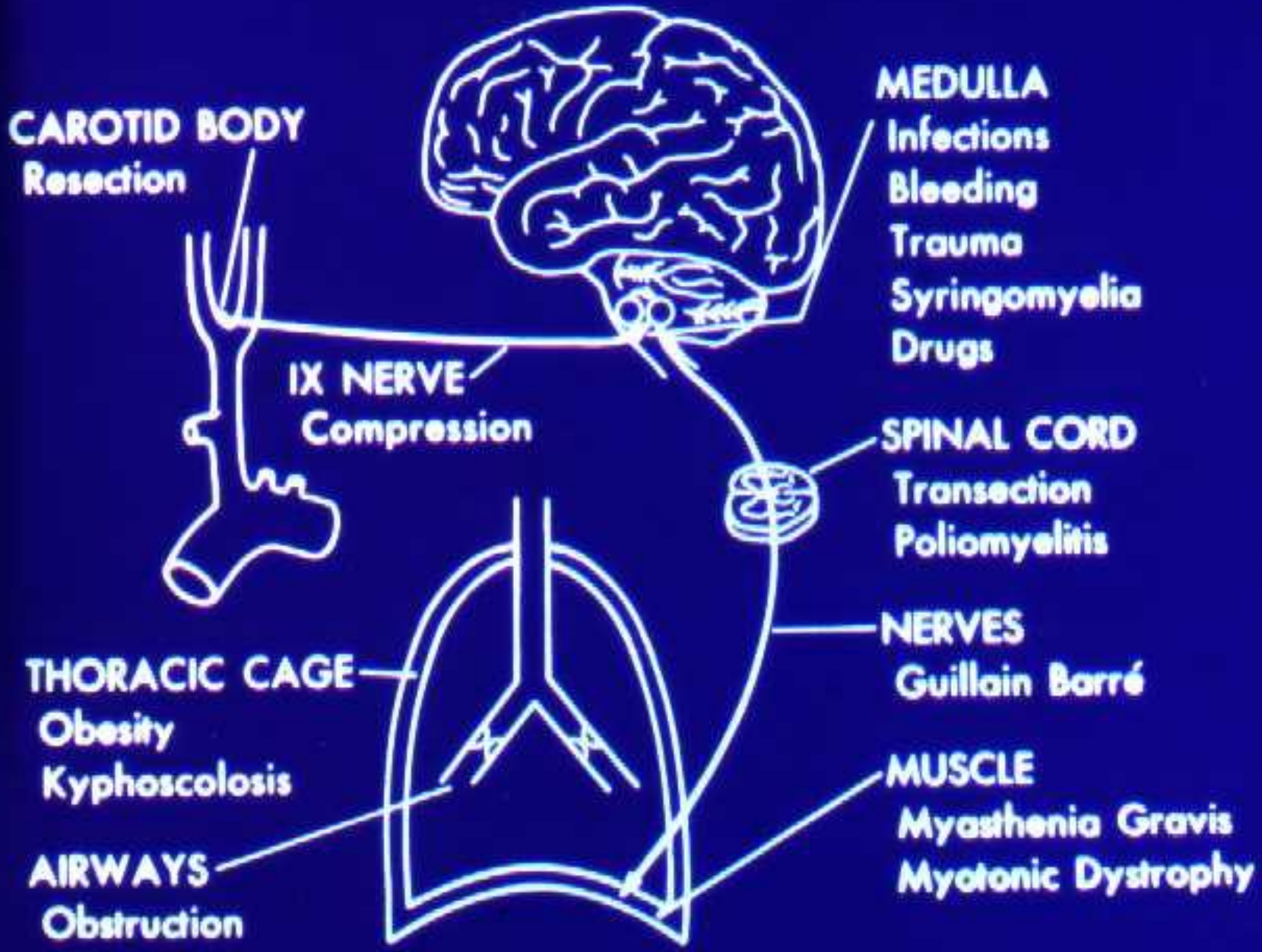
- Clinically

  Respiratory failure is defined as PaO_2 <60 mmHg while breathing air, or a $PaCO_2$ >50 mmHg.

Respiratory system includes:

CNS (medulla)
Peripheral nervous system (phrenic nerve)
Respiratory muscles
Chest wall
Lung
Upper airway
Bronchial tree
Alveoli
Pulmonary vasculature

Potential causes of Respiratory Failure



Pathophysiology

- type I respiratory failure
- When disease impairs ventilation of part of a lung (e.g. in asthma or pneumonia), perfusion of that region results in hypoxic and CO2- laden blood entering the pulmonary veins. Increased ventilation of neighbouring regions of normal lung can increase CO2 excretion, correcting arterial CO2 to normal, but cannot augment oxygen uptake because the haemoglobin flowing

Pathophysiology

through these normal regions is already fully saturated. Admixture of blood from the under-ventilated and normal regions thus results in hypoxia with normocapnia.

- type II respiratory failure
- it is seen in conditions that cause generalised, severe ventilation– perfusion mismatch, leaving insufficient normal lung to correct *Pa*CO2,

Pathophysiology

or any disease that reduces total ventilation. The latter includes not just diseases of the lung but also disorders affecting any part of the neuromuscular mechanism of ventilation.

HYPOXEMIC RESPIRATORY FAILURE(TYPE 1)

- PaO_2 <60mmHg with normal or low $PaCO_2$ $\rightarrow$ normal or high pH
- Most common form of respiratory failure
- Lung disease is severe to interfere with pulmonary O_2 exchange, but over all ventilation is maintained
- Physiologic causes: V/Q mismatch and shunt

HYPOXEMIC RESPIRATORY FAILURE
CAUSES OF ARTERIAL HYPOXEMIA

1. ↓FiO_2
2. Hypoventilation
   (↑ $PaCO_2$)
3. V/Q mismatch
   (eg.COPD)
4. Diffusion limitation ?
5. Intrapulmonary shunt
   - pneumonia
   - Atelectasis
   - CHF (high pressure pulmonary edema)
   - ARDS (low pressure pulmonary edema)

Hypercapnic
Respiratory failure

	Type I		Type II	
	Hypoxia (PaO_2 <8.0 kPa (60 mmHg)) Normal or low $PaCO_2$ (≤6 kPa (45 mmHg))		Hypoxia (PaO_2 <8.0 kPa (60 mmHg)) Raised $PaCO_2$ (>6 kPa (45 mmHg))	
	Acute	Chronic	Acute	Chronic
H^+	→	→	↑	→ or ↑
Bicarbonate	→	→	→	↑
Causes	Acute asthma Pulmonary oedema Pneumonia Lobar collapse Pneumothorax Pulmonary embolus ARDS	COPD Lung fibrosis Lymphangitic carcinomatosis Right-to-left shunts	Acute severe asthma Acute exacerbation of COPD Upper airway obstruction Acute neuropathies/paralysis Narcotic drugs Primary alveolar hypoventilation Flail chest injury	COPD Sleep apnoea Kyphoscoliosis Myopathies/muscular dystrophy Ankylosing spondylitis

(ARDS = acute respiratory distress syndrome; COPD = chronic obstructive pulmonary disease)

Causes of Hypoxemic Respiratory failure

- Caused by a disorder of heart, lung or blood.

- Etiology easier to assess by CXR abnormality:

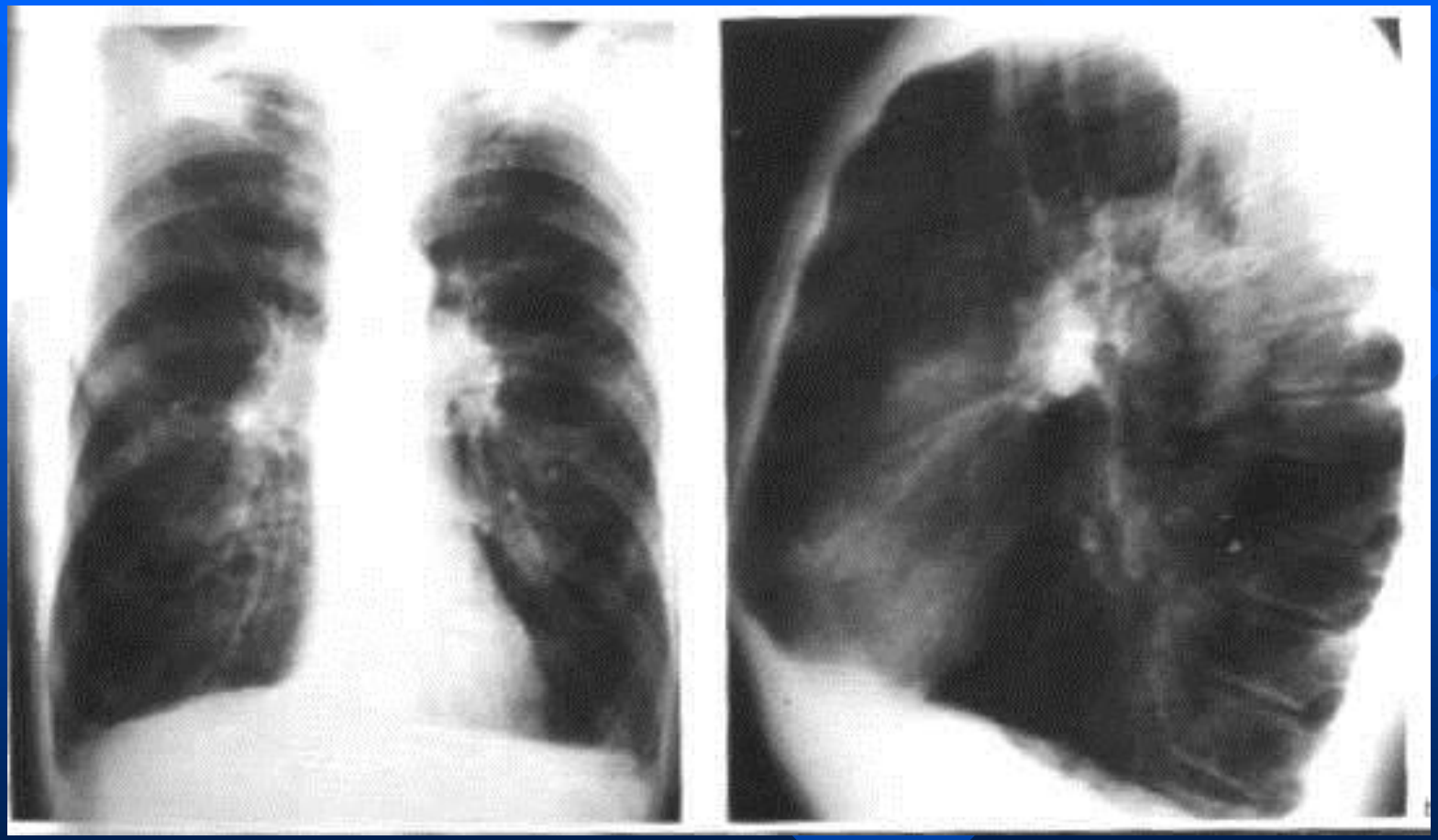
  - Normal Chest x-ray

    Cardiac shunt (right to left)

    Asthma, COPD

    Pulmonary embolism

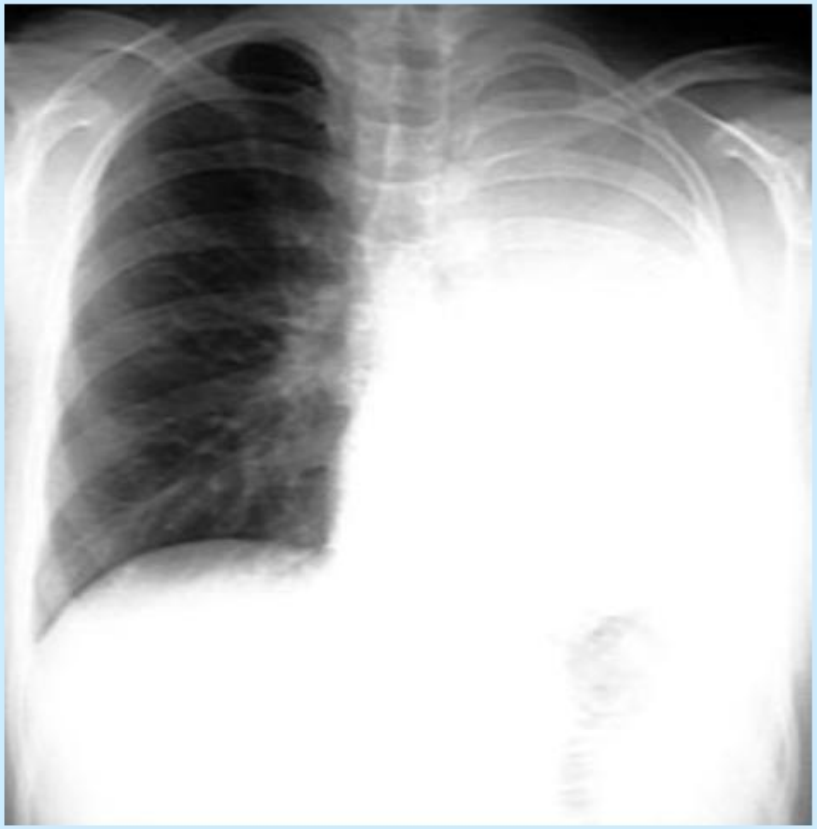
Hyperinflated Lungs : COPD



Causes of Hypoxemic Respiratory failure (cont'd.)

- Focal infiltrates on CXR
  - Atelectasis
  - Pneumonia

An example of intrapulmonary shunt

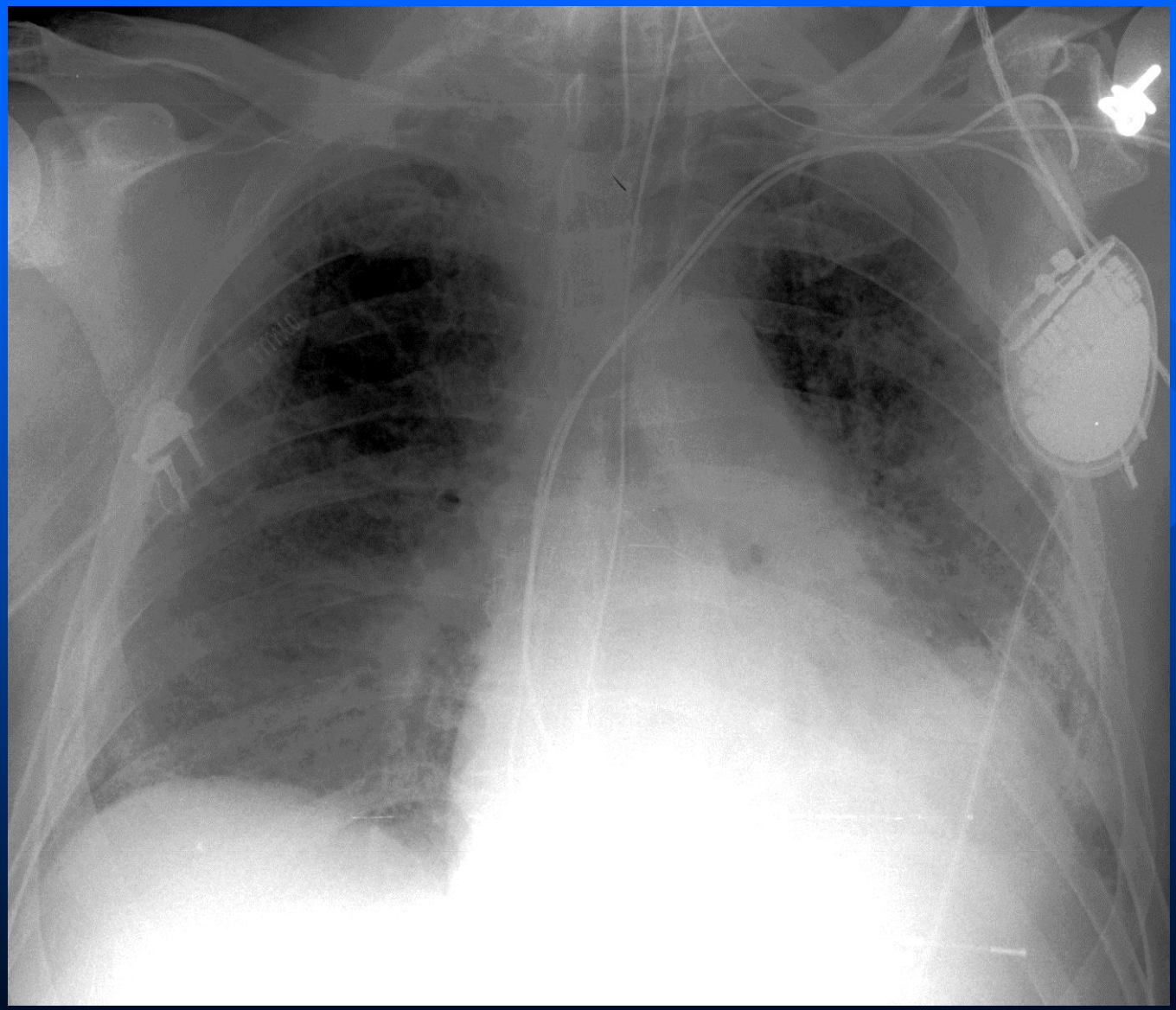


Causes of Hypoxemic Respiratory Failure (cont'd.)

Diffuse infiltrates on CXR

- Cardiogenic Pulmonary Edema
- Non cardiogenic pulmonary edema (ARDS)
- Interstitial pneumonitis or fibrosis
- Infections

Diffuse pulmonary infiltrates



Hypercapnic Respiratory Failure (Type II)

- $PaCO_2$ >50 mmHg
- Hypoxemia is always present
- pH depends on level of HCO_3
- HCO_3 depends on duration of hypercapnia
- Renal response occurs over days to weeks

Acute Hypercapnic Respiratory Failure (Type II)

- Acute
- Arterial pH is low
- Causes

  - sedative drug over dose
  - acute muscle weakness such as myasthenia gravis
  - severe lung disease:
    alveolar ventilation can not be maintained (i.e. Asthma or pneumonia)

- Acute on chronic:
- This occurs in patients with chronic CO_2 retention who worsen and have rising CO_2 and low pH.
- Mechanism: respiratory muscle fatigue

Causes of Hypercapnic Respiratory failure

- Respiratory centre (medulla) dysfunction
- Drug over dose, CVA, tumor, hypothyroidism,central hypoventilation
- Neuromuscular disease
  Guillain-Barre, Myasthenia Gravis, polio, spinal injuries
- Chest wall/Pleural diseases
  kyphoscoliosis, pneumothorax, massive pleural effusion
- Upper airways obstruction
  tumor, foreign body, laryngeal edema
- Peripheral airway disorder
  asthma, COPD

Clinical and Laboratory Manifestation

(non-specific and unreliable)

- Cyanosis
  - bluish color of mucous membranes/skin indicate hypoxemia
- - unoxygenated hemoglobin 50 mg/L
  - not a sensitive indicator
- Dyspnea
  - secondary to hypercapnia and hypoxemia
- Paradoxical breathing
- Confusion, somnolence and coma
- Convulsions

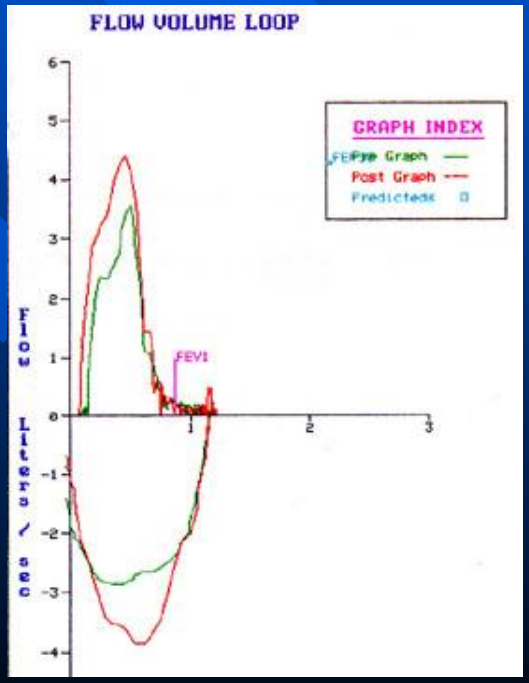
ASSESSMENT OF PATIENT

- Careful history
- Physical Examination
- ABG analysis
  -classify RF and help with cause

  1) $PaCO_2 = \frac{VCO_2}{VA} \times 0.863$

  2) $P(A\text{-}a)0_2 = (PiO_2 - \frac{PaCO_2}{R}) - PaO_2$

- Lung function
  OVP vs RVP vs NVP
- Chest Radiograph
- ECG



Clinical & Laboratory Manifestations

- Circulatory changes
  - tachycardia, hypertension, hypotension
- Polycythemia
  - chronic hypoxemia - erythropoietin synthesis
- Pulmonary hypertension
- Cor-pulmonale or right ventricular failure

Management of Respiratory Failure Principles

- Hypoxemia may cause death in RF
- Primary objective is to reverse and prevent hypoxemia
- Secondary objective is to control $PaCO_2$ and respiratory acidosis
- Treatment of underlying disease
- Patient's CNS and CVS must be monitored and treated

In type I respiratory failure

- High concentrations of oxygen (40–60% by mask) will usually relieve hypoxia by increasing the alveolar *P*O2 in poorly ventilated lung units.
- mechanical ventilation
- may be needed to relieve hypoxia. Occasionally, (e.g. severe pneumonia affecting several lobes
- humidified oxygen for Pts who need high concentrations for more than a few hours

Management

- Treat the cause
- Tension pneumothorax
  - Needle aspiration and chest drain
- Pulmonary oedema (diuretics)
- PE :Anticoagulant and thrombolytics
- Asthma ,COPD treatment

type II respiratory failure

- Acute type II respiratory failure
- is an emergency requiring immediate intervention
- It is useful to distinguish acute upper airway obstruction((rapid respiratory rate and accessory muscle recruitment with stridor)
  - Heimlich manoeuvre
  - immediate intubation or
  - Emergency tracheostomy

type II respiratory failure

- high-concentration (e.g. 60%) oxygen should be administered, pending a rapid examination of the respiratory system and measurement of arterial blood gases
- Treatment of COPD OR Asthma exacerbations
- Supported ventilation
  - is required if failure to respond to initial treatment, declining conscious level and worsening respiratory

Chronic and ‘acute on chronic’ type II respiratory failure

- The most common cause of chronic type II respiratory failure is severe COPD.
- These patients have lost their chemosensitivity to elevated *Pa*CO2, and so they may paradoxically depend on hypoxia for respiratory drive and are at risk of respiratory depression if given high concentrations of oxygen

‘acute on chronic’ type II respiratory failure

- some patients with ‘acute on chronic’ type II respiratory failure due to COPD may not appear distressed, despite being critically ill with severe hypoxaemia, hypercapnia and acidaemia. While the physical signs of CO2 retention (delirium, flapping tremor, bounding pulses and so on) can be helpful if present, they may not be, so measurement of arterial blood gases is mandatory in the assessment of initial severity and response to treatment

Assessment and management of 'acute on chronic' type II respiratory failure

Initial assessment

Patient may not appear distressed, despite being critically ill

- Conscious level (response to commands, ability to cough)
- CO_2 retention (warm periphery, bounding pulses, flapping tremor)
- Airways obstruction (wheeze, prolonged expiration, hyperinflation, intercostal indrawing, pursed lips)
- Cor pulmonale (peripheral oedema, raised jugular venous pressure, hepatomegaly, ascites)
- Background functional status and quality of life
- Signs of precipitating cause (see Box 17.15)

Investigations

- Arterial blood gases (severity of hypoxaemia, hypercapnia, acidaemia, bicarbonate)
- Chest X-ray

Management

- Maintenance of airway
- Treatment of specific precipitating cause
- Frequent physiotherapy ± pharyngeal suction
- Nebulised bronchodilators
- Controlled oxygen therapy:
  - Start with 24% Venturi mask
  - Aim for a $PaO_2 > 7$ kPa (52 mmHg) (a $PaO_2 < 5$ (37 mmHg) is dangerous)
- Antibiotics if evidence of infection
- Diuretics if evidence of fluid overload

Progress

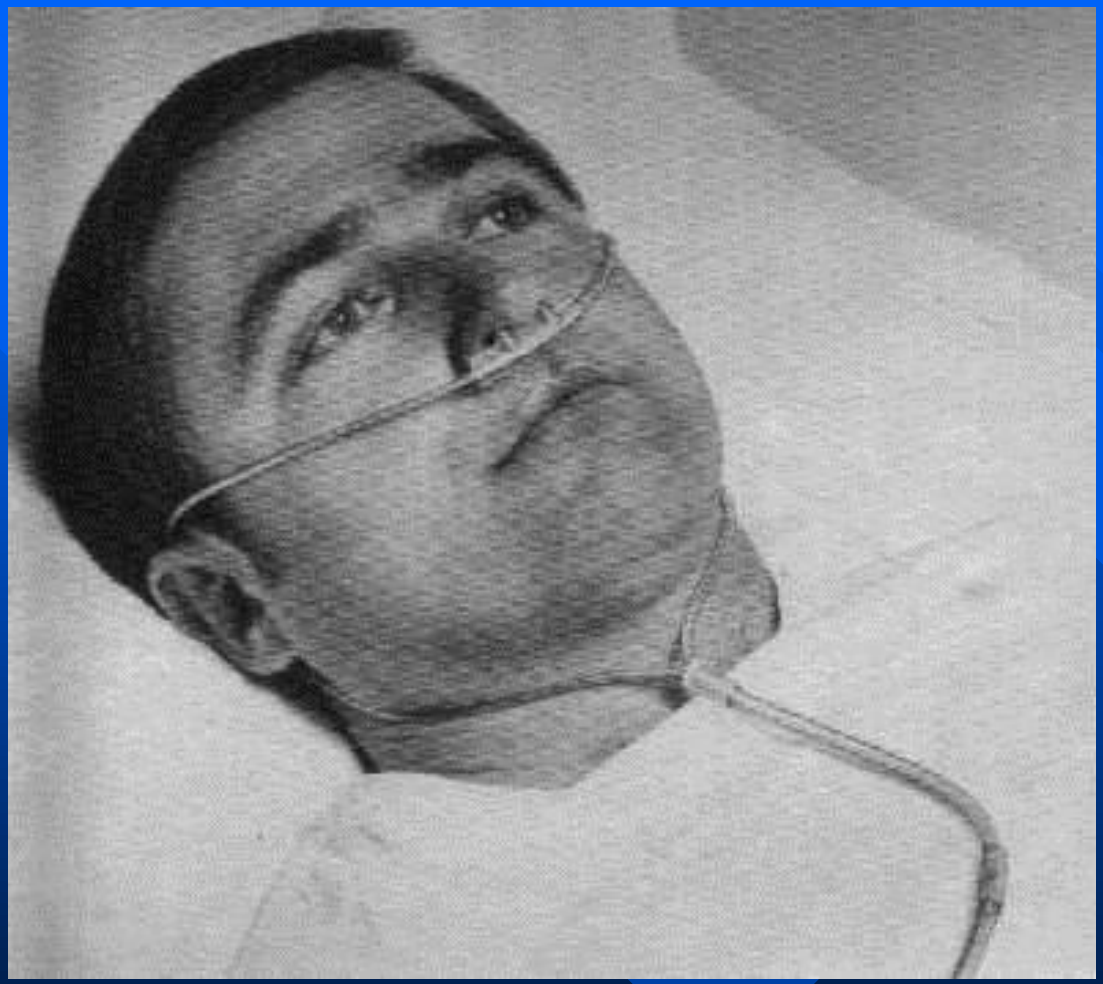
- If $PaCO_2$ continues to rise or a safe PaO_2 cannot be achieved without severe hypercapnia and acidaemia, mechanical ventilatory support may be required

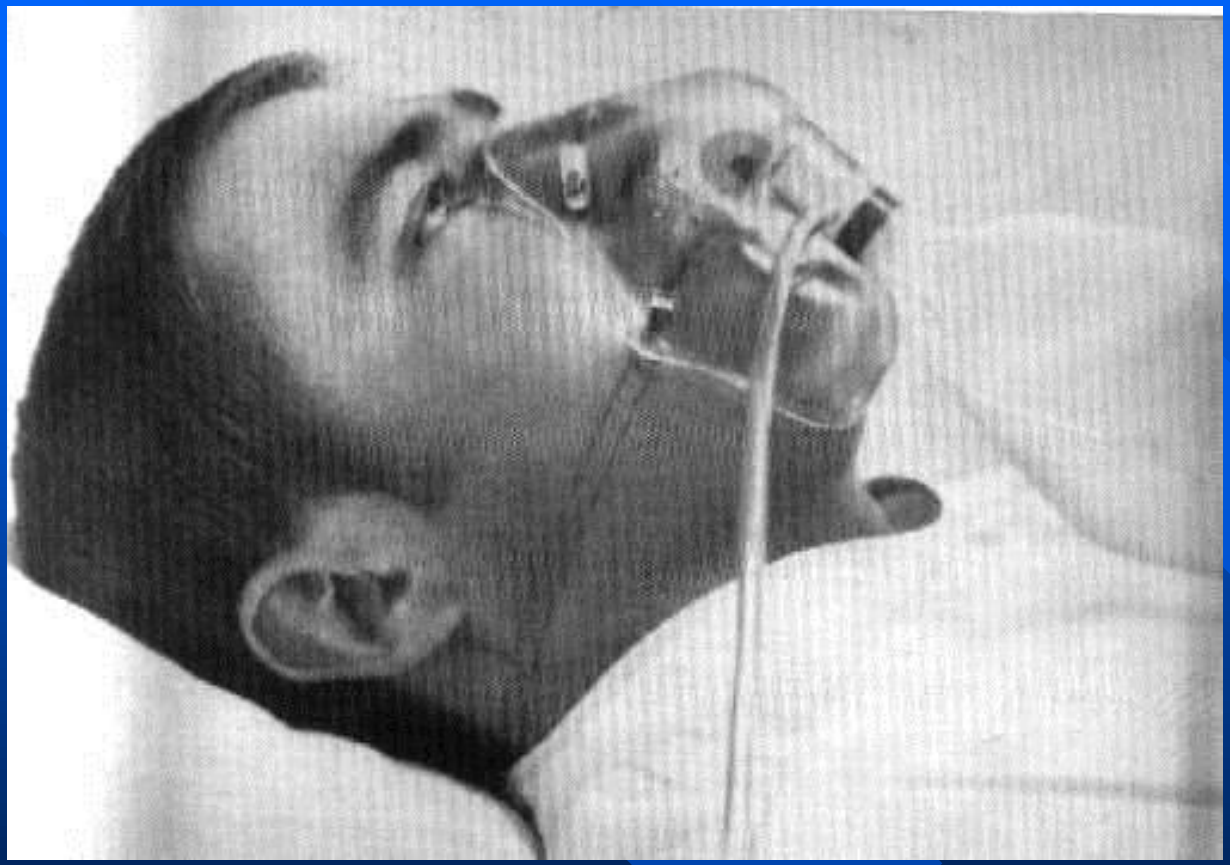
Oxygen Therapy

- Supplemental O_2 therapy essential
- Titration based on SaO_2, PaO_2 levels and $PaCO_2$
- Goal is to prevent tissue hypoxia
- Tissue hypoxia occurs (normal Hb & C.O.)
  - venous $PaO_2 < 20$ mmHg or $SaO_2 < 40\%$
  - arterial $PaO_2 < 38$ mmHg or $SaO_2 < 70\%$
- Increase arterial $PaO_2 > 60$ mmHg($SaO_2 > 90\%$) or venous $SaO_2 > 60\%$
- O_2 dose either flow rate (L/min) or FiO_2 (%)

Risks of Oxygen Therapy

- **O_2 toxicity**:
  - very high levels(>1000 mmHg) CNS toxicity and seizures
  - lower levels (FiO_2 > 60%) and longer exposure: - capillary damage, leak and pulmonary fibrosis
  - PaO_2 >150 can cause retrolental fibroplasia
  - FiO_2 35 to 40% can be safely tolerated indefinitely
- **CO_2 narcosis:**
  - $PaCO_2$ may increase severely to cause respiratory acidosis, somnolence and coma
  - $PaCO_2$ increase secondary to combination of
    a) abolition of hypoxic drive to breathe
    b) increase in dead space





MECHANICAL VENTILATION

- Non invasive with a mask
- Invasive with an endobronchial tube
- MV can be volume or pressure cycled
  For hypercapnia:
  - MV increases alveolar ventilation and lowers $PaCO_2$, corrects pH
  - rests fatigues respiratory muscles
- For hypoxemia:
  - O_2 therapy alone does not correct hypoxemia caused by shunt
  - Most common cause of shunt is fluid filled or collapsed alveoli (Pulmonary edema)

POSITIVE END EXPIRATORY PRESSURE (PEEP)

- PEEP increases the end expiratory lung volume (FRC)
- PEEP recruits collapsed alveoli and prevents recollapse
- FRC increases, therefore lung becomes more compliant
- Reversal of atelectasis diminishes intrapulmonary shunt
- Excessive PEEP has adverse effects
  - decreased cardiac output
  - barotrauma (pneumothorax, pneumomediastinum)
  - increased physiologic dead space
  - increased work of breathing

Adult Respiratory distress Syndrome (ARDS)

- Variety of unrelated massive insults injure gas exchanging surface of Lungs
- First described as clinical syndrome in 1967 by Ashbaugh & Petty
- Clinical terms synonymous with ARDS
  - Acute respiratory failure
  - Capillary leak syndrome
  - Da Nang Lung
  - Shock Lung
  - Traumatic wet Lung
  - Adult hyaline membrane disease

Risk Factors in ARDS

Sepsis	3.8%
Cardiopulmonary bypass	1.7%
Transfusion	5.0%
Severe pneumonia	12.0%
Burn	2.3%
Aspiration	35.6%
Fracture	5.3%
Intravascular coagulopathy	12.5%
Two or more of the above	24.6%

PATHOPHYSIOLOGY AND PATHOGENESIS

- Diffuse damage to gas-exchanging surface either alveolar or capillary side of membrane
- Increased vascular permeability causes pulmonary edema
- Pathology: fluid and RBC in interstitial space, hyaline membranes
- Loss of surfactant: alveolar collapse

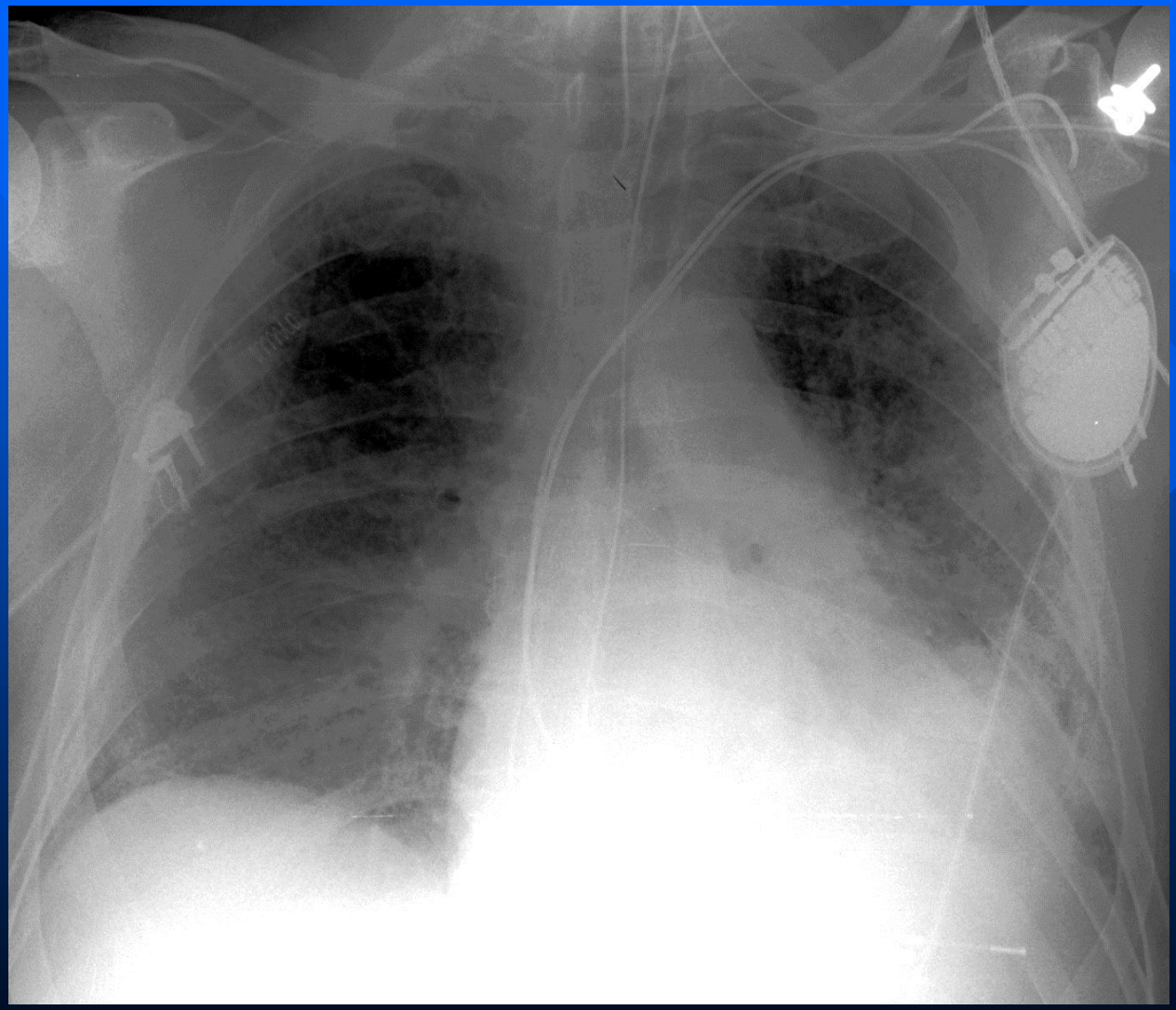
CRITERIA FOR DIAGNOSIS OF ARDS

- Clinical history of catastrophic event
  - Pulmonary or Non pulmonary (shock, multi system trauma)
- Exclude
  - chronic pulmonary diseases
  - left ventricular failure

Must have respiratory distress

- tachypnea >20 breath/minute
- Labored breathing
- central cyanosis
- CXR- diffuse infiltrates
- PaO_2 <50mmHg FiO_2 >O.6
- Compliance <50 ml/cm H_2O increased shunt and dead space

ARDS



MANAGEMENT OF ARDS

- Mechanical ventilation (preventive strategy)
  corrects hypoxemia/respiratory acidosis
- Fluid management
  correction of anemia and hypovolemia
- Pharmacological intervention
  Dopamine to augment C.O.
  Diuretics
  Antibiotics
  Corticosteroids - no demonstrated benefit
  early disease, helpful 1 week later

Others treatment

- Prone position
- ECMO
- Mortality
- continues to be 50 to 60%

THANK YOU